VOL. XXVIII. April, 1901. No. 4.

THE Journal OF Nervous and Mental Disease.

Original Articles.

THE CLINICAL VALUE OF ASTEREOGNOSIS, AND ITS BEARING UPON CEREBRAL LOCALIZATION.[1]

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Astereognosis has become established as an important symptom to be investigated in all cases at least of suspected cerebral disease. Many such cases are deemed normal as regards sensation, in which this investigation would not only furnish material aid in diagnosis but also further the study of cerebral localization. The importance of the subject has been recognised by various observers, to whose contributions (in which the expression *active touch* is more often used), reference is made in the exhaustive work of Verger[2] on Cerebral Anesthesia. The study was given an impetus in this country by the work of Dana,[3] and the importance of testing for astereognosis has been emphasized in Philadelphia by the contributions of Dercum,[4] Burr[5] and Sailer,[6] as well as by the practical application in diagnosis and treatment shown in the brilliant case of Mills and Keen.[7]

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[1] Read by invitation before the Philadelphia Neurological Society, February 25, 1901.

[2] Archives Général de Médicine, Nov. and Dec. 1900.

[3] Journal of Nervous and Mental Disease, October 1898, and December 1894.

Complete hemianesthesia involving the trunk to the median line and including analgesia, whether the special senses are affected or not, generally means hysteria. In fact, complete anesthesia leaving off abruptly at a point short of the median line, for example, at the shoulder, is apt to fall in the same category. This variety of anesthesia is rarely to be confounded with that of organic origin. In hysteria the character of the astereognosis as well as the distribution of anesthesia and its completeness, will generally indicate the nature of the malady. Such patients, for example, while taking no notice of pinch or prick, and stating that no object is recognised when the stereognostic sense is tested, will handle objects naturally and freely with the affected hand. One such patient coming recently under our observation, through the courtesy of Dr. Shattuck, stated that no touch of any kind was felt up to the shoulder, and failed to recognise objects placed in the hand. She found no difficulty, however, in extracting her handkerchief from the recesses of her clothing, though it was concealed from view. This indication of subconscious recognition we have not found in astereognosis of organic origin. True cerebral anesthesia is recognised by its distribution and by its character. Its distribution is distal, that is, either limited to the extremities, more particularly to the hands and feet, or if involving also the trunk, the anesthesia shades off gradually as the middle line is approached. With regard to the character, the loss is generally more marked in the posture, spacing and localizing senses than in those of touch, pain, and temperature. In certain of these cases the loss of pain sense is also marked, and to this class Verger has applied the name pseudo-hysterical.

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[4] Journal of Nervous and Mental Disease, August 1899, and November 1900.

[5] Journal of Nervous and Mental Disease, May 1897, and January 1898.

[6] Journal of Nervous and Mental Disease, March 1899.

[7] Journal of Nervous and Mental Disease, May 1900. University Medical Magazine, October 1897.

An object placed in the affected hand of a patient with cerebral anesthesia is unrecognised if the eyes are closed—that is, the stereognostic sense (or sense of "active touch") is lacking. For brevity we will use the term astereognostic type.

Cerebral anesthesia of the astereognostic type occurs in both cortical and central disease, between which we are unable to distinguish by this symtom alone as shown by the studies of Dejerine,[8] and Long. With the aid furnished in many cases by hemianopsia on the one hand, and by monoplegic distribution and Jacksonian epilepsy on the other, we have been long familiar, but it was to be hoped that we might add another factor of diagnostic value. Such cases, however, as that of Long[9] seem conclusive on this point—the autopsy here revealed extensive softening of the posterior two-thirds of the internal capsule, but no sign of cortical disease. In this case touch, temperature, and pain senses were normal, but muscle and position senses were lost.

This case also serves as a reminder of the fact, to which further reference will be made, that the muscle sense fibres probably follow a separate course from those conveying cutaneous sensations. The curious fact that in Sailer's second case a deep lesion causing hemianopsia and pseudo-hysterical anesthesia on one side, was accompanied by the pure astereognostic type on the other, while hard to explain satisfactorily, certainly tends to show that cortical lesion is not essential to the latter form of anesthesia.

An interesting example of cerebral anesthesia resulting apparently from interruption of the bulbo-thalamic fibers recently came under our observation in the Neurological Department of the Massachusetts General Hospital, and will be elsewhere reported in detail by Dr. Paul.[10]

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[8] Semaine Médicale, July 20, 1899.

[9] Thèse de Long, p. 246, cited by Verger.

[10] Notes from the Neurological Department of the Massachusetts General Hospital, Boston Med. and Surg. Journal, March 14, 1901.

Case I.—This patient, a woman 51 years of age, presented typical bilateral labio-lingual palsy, of ten years' duration, and of apoplectiform onset. The bulbar symptoms were accompanied by weakness and numbness of the left hand. She is unable to-day to recognize objects placed in this hand. There is very slight impairment of touch and pain sense extending up the arm, gradually disappearing after passing the shoulder. There is no loss of temperature, localizing, or posture sense, but slight impairment of space sense. Aside from the question of astereognosis this case presents a clinical picture of great interest, and, in fact, rarity; such cases are, however, mentioned by Gowers under the head of "Sudden (Apoplectiform) Bulbar Paralysis."

It seems fairly established that the ability to recognise objects held in the hand with the eyes closed, is a matter not of any one simple sensation, but a complex judgment, based on correlated memories of various forms of contact sense, spacing, localizing, pressure, together with the co-called muscle sense. With regard to the term muscle sense, the knowledge of the position of the members is imparted not only by stimuli from the muscles but also by cutaneous and articular stimuli; the former predominate when the parts are actively moved, the latter when they are at rest, but we can in neither case feel sure that we are only testing one, for even when at rest the muscles are in a state of tonus. It seems therefore advisable to use the term *posture sense* in referring to the patient's ability to recognise the direction in which the fingers, for example, are moved. In this way we merely record facts without misleading inferences. Pain and temperature senses are of little importance in this connection, and are apt to remain unaffected, except in matters of judgment, *e.g*, in localizing the stimulus. The preservation, or comparative preservation, of touch sense is the key to the situation, for loss of this sense alone would of course entail loss of power to recognize objects.

It would seem that in certain physiological groups of fibers and cells are aggregated the associative memories concerning the sensations produced by the various qualities of objects, and to these groups we doubtless appeal for the reproduction of the ideas previously aroused by such sensations. Such processes rank high in the psychical scale, and must involve highly differentiated association fibers and cortical cell groups. It is apparently in cortical or subcortical disease that astereognosis is likely to prove of material diagnostic assistance, though it is not pathognomonic of this limitation.

In the second case we have to report, astereognosis was the initial, and for an appreciable period the only localizing symptom. This alone was deemed sufficient to justify a probable diagnosis of subcortical lesion, a diagnosis subsequently established by operation.

Case II. Nellie B.—This young woman presented herself at the clinic, Neurological Department of the Massachusetts General Hospital, Friday, November 17, 1900, complaining of numbness in the left arm and leg. The symptom had been of gradual onset, commencing in the leg about six weeks earlier, ushered in by temporary blur of vision and loss of power. The history included slight occipital headache, and occasional involuntary movement of the left hand, not amounting to spasm. There was a slight awkwardness in the left hand, but the separate motions were made perfectly and with good strength; the grasps were practically alike. The knee-jerk was exaggerated on the left. Ankle-clonus was present, but there was no Babinski reflex.

In other respects she was well, and doing her work as a compositor. It was found that when objects were placed in the left hand, she was unable to name them with closed eyes, for example, a knife, a coin, a pencil. On analysing the anesthesia only a slight degree of loss of touch sense appeared. The temperature and pain senses were preserved. Posture sense was lost; she could not tell with the eyes closed whether the fingers were raised, lowered, or separated. Examination was otherwise negative, including that of the fundus oculi. It was decided not to advise operation until the symptoms became more marked. Monday she returned, when Dr. Putnam examined her and agreed in the diagnosis.

The patient continued her work and was practically well up to Wednesday, when there was rapid onset of left hemiplegia, violent headache and vomiting. Dr. Hamilton attended the patient at her home, and Drs. Putnam and Lord were called to see her. Her condition steadily grew worse and she was urged to enter the hospital for operation. She delayed until Friday morning, when her general condition had become alarming. At this time the Babinski reflex was present on the left. The operation was performed that day by Dr. Warren.

Operation. It was planned that the center of the flap should fall just posterior to the fissure of Rolando at the height of the moter centers of the arm.

With regard to the technique of operation, two trephine holes well separated were made in the right parietal region and connected by cutting forceps. The flap was completed by two converging lines made by the same instrument, and the narrowed base fractured and reflected with its covering of skin. The meningeal flap was laid back in the opposite direction as suggested by Keen. The surface of the brain revealed no abnormality by inspection or palpation beyond a moderate degree of tension. Introduction of the trocar at the point indicated and directed toward the median line gave vent, however, to about a testtubeful of clear fluid, examination of which was negative. The opening was enlarged and the cyst wall explored without sufficiently definite results to demand further operation. The dura was sutured and the bone and skin flap replaced, with drainage.

Uninterrupted improvement took place for three weeks, during which time only occasional moderate headache was complained of, and the paralysis was gradually lessening. Occasional tests showed loss of posture and space senses, impairment of pressure sense, moderate blunting of touch, with preservation of pain and temperature senses. At this time we confirmed the observation first made by Long in 1896, that when such a patient is told with the eyes closed to touch the affected hand with the other it is found impossible to do so except after groping. This indicates, of course, complete lack of posture sense in the affected part.

At the end of three weeks headache and vomiting recurred and grew steadily worse, the patient failed, and in one week died after a slight convulsion. Autopsy was refused.

This case presents astereognosis in its typical form. The following case falls in the same category, excepting that the loss of stereognostic sense was less in degree.

Case III.—J. W., a young woman, presented herself at the Neurological Department of the Massachusetts General Hospital, December 7, 1900, with the following history. She was well previous to November 1, when she noticed numbness of the right hand while washing dishes. The numbness spread to the elbow and in a few days loss of power in the hand and whole arm followed. Several days later she lost power, and at the same time sensation, in the right leg. About this time her speech became affected. Moderate headache and vomiting appeared early in the history, but of late had subsided.

Physical examination showed spastic condition of the right leg with Babinski reflex but no clonus. The knee-jerk was exaggerated on both sides, but more marked on the right. Movements of the right hand were awkward. It seemed hard for the patient to find words but there was no decided aphasia. The fundus was normal. (Dr. Cheney).

Examination of the hand with reference to stereognostic sense gave the following results:—at the first trial the patient could not distinguish between a round and a square object, nor name a quarter dollar piece, a knife, a pencil, a piece of paper, or a key, but she recognised a bottle. At the second trial all of these objects were recognised, excepting the key.

Touch sense was found normal, the patient answering quite promptly each time she was touched. Pain and temperature senses were normal. Pressure sense, tested by variously weighted bottles of the same size, was lessened. Posture sense was moderately impaired, the patient at one time answering correctly as to the position of the fingers, her answers at another being incorrect. The localizing sense, normal in the palm, was lost in the finger tips, the patient being unable to state which finger was touched. Space sense was blunted; two points were named one until widely separated, after which all applications were named two, both when the points were closely approximated and when only one point was applied.

The patient was referred to the wards, entering the service of Dr. Fitz. While tumor was deemed the probable diagnosis, the rapid onset of symptoms together with the subsidence of vomiting and headache, and the absence of optic neuritis, prevented an absolute diagnosis. It was thought advisable to keep the patient under close observation so as to be prepared for operation whenever the diagnosis of tumor should be established, and, if possible, before alarming symptoms should supervene.

The condition remained practically unchanged until the last of the month when headache and vomiting recurred, double optic neuritis appeared, the paralysis extended to the face, and the general condition became worse. With the concurrent opinion of Drs. Fitz and Putnam, immediate operation was advised, and the patient was transferred to the surgical wards.

Operation was performed by Dr. H. H. A. Beach, January 1, 1901. A flap three by three inches over the Rolandic region of the left side was turned back including the skull with the skin, the former being opened by the rongeur forceps and cutters starting from a ¾-inch trephine hole. The dura was opened revealing a dark, discolored area of the cortex the size of a half dollar, behind the fissure of Rolando. A small fragment of the cortex removed for examination was pronounced by Dr. Fitz malignant. It was deemed inadvisable to attempt removal. Another piece examined later by Dr. Wright was pronounced free of neoplasm. Introduction of a hollow needle was negative. The bone was removed from the flap to insure relief from pressure, and the skin was closed without drainage by interrupted silk-worm gut sutures. The post-operative condition was poor, the pulse 150. The condition improved on the second day. No change in the paralysis developed excepting that aphasia became marked, the patient being able to use only the words, "yes "and "middling." The aphasia improved from day to day through the first week, since which time it has remained stationary. When the aphasia was at its worst word-deafness and mind-blindness were marked, the patient understanding only the simplest orders and being unable to imitate even the simplest motions, as showing the teeth. A large cerebral hernia appeared at the seat of operation. The pressure symptoms disappeared. The sensory aphasia has prevented further examination of the stereognostic sense. No material change in her condition appeared up to the time of her discharge at the end of the month.

The extension of the post-operative symptoms in this case illustrates the danger, to which Mills has alluded, of drawing too definite conclusions from single cases of this nature.

The next case, first seen in December, 1900, and transferred to the wards, entering Dr. Shattuck's service, is of interest in that the paralysis was limited to the lower extremity, and furnished opportunity to demonstrate the analogue in the foot of astereognosis in the hand.

With a view to determining the normal degree of accuracy in the contact sense of the foot, we made a series of observation on healthy individuals. The result would indicate that in health we can do little more than determine whether the object touching the foot is sharp, blunt or long. In the persons tested, no reliability was to be placed on their discrimination between the touch of the finger, of the open end of a test tube, and of a quarter dollar piece. No difficulty was found, however, in distinguishing objects of this nature from a long object, as a pencil applied transversely to the sole. The latter test was found to suffice for practical purposes in the class of cases under consideration. It was found, for example, that a typical hemiplegic with marked astereognosis in the right hand found it impossible to distinguish between a quarter dollar piece and a pencil even when the latter was applied transversely to the sole of the paralysed foot. Localizing, space, and posture senses were found practically wanting in the foot of this patient, the first two being of course the only factors entering into the test.

Allusion has been already made to Long's test of the patient's inability to touch the astereognostic hand with the other unaided by vision. Trial of this capacity in the normal lower extremity showed that designated points upon one leg or foot could be located quite as accurately (and more conveniently) by the toe or heel of the other foot, as by the finger. This method is especially applicable to patients in bed.

Case IV.—Mrs. A. C., 27 years old, had a convulsion two years ago followed by paralysis of the left leg. This paralysis gradually became more marked with spastic symptoms; she had had repeated epileptiform attacks of Jacksonian type beginning in the great toe, recurring of late two or three times a week. After the attacks there was temporary weakness and awkwardness of the left arm. The face was unaffected. Headache and vomiting supervened in the past seven or eight weeks. There was double optic neuritis, the knee-jerks were increased, there was ankle-clonus on the left with Babinski reflex. No astereognosis existed in the hand.

The sensation was carefully tested in the paralysed foot, and it was found that though touch-sense was preserved, she was unable to distinguish between the quarter dollar piece and the pencil applied transversely to the left sole, though perfectly able to do so on the right. The temperature and pain senses were practically normal, though certain tests tended to corroborate the statement that the judgment may be faulty though thermic stimuli are recognized, *e.g.*, though she readily told the difference between hot and cold test-tubes on the affected side, when the same tube was applied to both extremities she called it colder on one side than on the other. Posture sense was lost in the toes, spacing sense was wanting, two points being felt as one 6 c.m. apart, that is, separated the entire width of the foot. On the other foot these senses were preserved. The movements of the left foot were so ataxic that it was impossible for the patient to touch the finger of the examiner with her toe. She was unable to touch the toe or the heel of the left foot with the toe of the other foot without groping, whereas the knee was readily located.

Careful analysis of the loss of posture sense in this case, as in the others, shows that even in this sphere we have to do rather with loss of judgment than with loss of sense. This patient, *e.g.*, never failed to recognize the fact that the position of her toe was changed, though she was totally unable to state the direction of the change. We should be chary, however, of drawing conclusions as to the true muscle sense, for her recognition might be due to cutaneous or articular impressions.

Operation. On January 16, 1901, the patient was transferred to the surgical wards in the service of Dr. Warren, who operated January 22. Having in mind the center for the great toe (the initial point of attack in the convulsions) a point was chosen just posterior to the upper end of the fissure of Rolando as the center of the area to be exposed. A large flap of skin and bone was turned down. The section of bone was made by the cutting forceps with a three-quarter inch trephine hole near the median line as a starting point. The dura was tense and very thin. Before cutting the dura, the opening was enlarged by rongeur forceps in its upper and posterior portion. On exposure of the brain the region anterior to the fissure of Rolando appeared normal, whereas nearly the entire surface exposed behind the fissure was discolored, bulged excessively, was extremely vascular, and contained a hemorrhagic focus, from which an old clot was removed as large as the end of the thumb. On account of the character and indefinite boundaries of the diseased area nothing further was done beyond removing a portion of the cortex.

A bit of cortex was examined at the time by Dr. Wright who found that the specimen submitted to him consisted of hemorrhagic cerebral tissue; he found no neoplasm, but stated that the condition was not inconsistent with tumor deeper in. A larger piece was examined later by Dr. Whitney, who reported as follows: "A mass of small, soft fragments, more or less reddish in color. Fresh examination showed the presence of nerve fibers and neuroglia. Microscopic examination of the hardened specimen showed small, dilated and thrombosed vessels with blood extravasated into the normal brain tissue. Nowhere in the specimen received was there any evidence of a new growth. Thrombosis of the small vessels and miliary foci of hemorrhage (red softening.)"

The exact nature of the lesion cannot, therefore, be determined, but the history, together with the pathological finding strongly suggests a highly vascular neoplasm. The next day the motor paralysis had extended to the hand, which now showed marked astereognosis. Objects at once named in the right hand were unrecognised in the left. The patient's general condition precluded detailed analysis of the anesthesia. It was noted, however, that with eyes closed she could not find the left arm with the right hand.

On the third day (January 26) she had recovered some movement in the arm and a little in the fingers. She declared herself free from headache, and feeling better than she had for a long time. Examination at this time showed in the left hand loss of posture and space senses, with preservation of temperature sense and slight blunting of pain and touch senses. Objects placed in the left hand with the eyes closed were unrecognised. It was noted at this time that the patient not only failed to recognise the direction in which the parts were moved, but she did not even know they were moved at all; this was true not only of the fingers but of the wrist. Eleven days after operation convulsive movements appeared in the left arm and leg without loss of consciousness. Up to the date of her discharge, about a month after the operation, no further attacks appeared. At this time the paralysis was more marked in the arm than in the leg.

The result of operation in these three cases opens up the broad question of the advisability of surgical interference in cerebral disease. Without entering into a discussion of this phase of the subject, we still feel that in spite of continued disappointments, an occasional successful operation like that of Mills and Keen, renders it our duty in these desperate cases to offer the patient the benefit of the chance when we have located the lesion at or near the cortex.

In approaching the subject of *astereognosis from a physiological point of view*, it would seem that there are certain factors the correlated memories of which enable us to determine the size and shape of objects. These factors have been analysed and catalogued in a manner somewhat arbitrary, perhaps, but sufficiently accurate for practical purposes. The different senses may be classified as (a) touch, (b) temperature, (c) pain, (d) posture, (e) localizing, (f) pressure, and (g) space. Astereognosis has been coincident in different cases with varying degrees of impairment of a varying number of these factors, and one case has been reported by Sailer and one by Burr tending to show that astereognosis may occur without loss of any other form of sensation.

It appears that in the typical case, certain forms of sensation play a major part, though perhaps the integrity of all is necessary for the very highest form of stereognosis. *A priori*, the most important senses would be those of spacing and localizing, which enable us to determine the size and shape of the surface or surfaces coming in contact with the skin; next in importance, posture sense tells the position of the fingers and their changes of movement in clasping and handling the object. Next would perhaps come the pressure sense through which the weight of the object is determined; of less importance, but still of aid, the temperature sense tells something of the material in certain cases; and of still less importance, pain sense indicates that the object possesses such extreme sharpness or excessive temperature as to produce discomfort. At the end of the scale comes touch sense, a sense possessing no special value in determining the size and shape of objects, though necessary for such determination.

It appears, on the whole, that the facts determined by the various observers of astereognosis substantiate this expectation.

It is, perhaps, not unreasonable to assume that the impressions of simple touch, pain, and temperature not only have extensive cortical representation, but are also recorded for the simpler reflex purposes, though not consciously, in centers more deeply seated, principally in the thalamus. Whatever the exact localization of these centers, the mechanism concerned is already developed far below man in the animal scale. Such animals are capable of recognising the fact that they are touched, of knowing when they are hurt, and of distinguishing between heat and cold. That they possess to a certain degree the associative memories which take part in stereognosis is evident from the fact that they can localize their sensations, but that they possess the complicated mechanism required for the perfected stereognostic sense is highly improbable. Experiments upon animals will be, therefore, of little aid in this study.

Posture sense, which, as we have used the term, must include muscle sense, may be regarded as a specialization of what is known as common sensation, that is, the sensation which acquaints us with the condition and position of our bodies. But while common sensation is probably of extensive cortical as well as of deep representation and of low order, giving rise, for example, to the ordinary reflex processes of changing the posture and maintaining the equilibrium, still the accuracy of this sense possessed by man, represents a high degree of education, requiring judgment and training and justifying its classification with the localizing and spacing senses.

Bastian[11] in 1887 suggested the possibility that the fibers conducting muscle sense to the cortex follow a separate course, and end in a special region. This region he placed in the central convolutions, while adopting Ferrier's view of cutaneous sensation. Experimental researches are corroborrative of this suggestion, and show, as indicated in the critical review of the subject by Barker,[12] (1) that fibers passing from the nuclei of the dorsal funiculi through the medial lemniscus convey muscle sense only, (Tschermak, von Bechterew, Ferrier and Turner); and (2) that these fibers pass in large part to the central gyri, either directly, (Flechsig and Hoesel), or with interruption, chiefly in the thalamus, (Mahaim, von Monakow). It would not be surprising if further study should accentuate this differentiation, but with this branch of the subject we shall not here concern ourselves, though it will be referred to in the tentative suggestions we shall offer regarding the localization of the stereognostic sense.

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[11] Brain, April, 1887.

[12] "The Nervous System and Its Constituent Neurones," Appleton, 1899, p. 697.

The experiments of Sailer show that even in man the so-called muscle sense is less reliable than contact sense, a combination of tactile, pressure, and localization sensations. We have tried experiments similar to those of Sailer on normal individuals with practically the same result. Out of twenty individuals tested, only four easily and invariably distinguished by approximating the finger and thumb, the difference in diameter between a quarter dollar and a five cent piece; of these four, three were physicians with long practice in the use of delicate instruments (two aurists and one laryngologist), and one was a gentleman of unusual manual dexterity. The latter in fact, never failed to distinguish the cent piece from the nickel. This test is more accurate when the coins are replaced by pieces of cardboard of the same diameter, and when this precaution was taken the successful percentage was still further reduced. When we remember how readily the average individual can distinguish even the cent piece from the nicked in his pocket the inadequacy of the posture sense becomes evident. Not only in this direction but in others, the study of stereognosis furnishes constant surprises; it was found that the open end of a test tube was not always distinguished in health from a solid object of the same diameter applied to the palm of the hand. Control observations are particularly important in this branch of physical examination.

With regard *to the localization of the areas concerned in astereognosis*, Verger states that he knows of no case in which cerebral anesthesia existed without motor paralysis. This occurred, however, in the case of Mills and Keen, and was adduced by the former to corroborate his view that the centers of sensation are distinct from those of motion. In the second and third cases reported in this paper a history was given which agrees with this experience, and in one of these cases we can confirm this history by own observation, for the astereognosis was fully established when the patient came to the clinic, and when no paralysis of motion existed. The rapid extension of symptoms, however, to include the motor functions in each of these cases forcibly suggests a close relationship in point of location between the motor centers and the highest areas subserving the stereognostic sense. The fact that with the extension of paralysis to the hand in the fourth sense moderate blunting of other senses occurred might be explained either on the theory that other senses are also represented in the Rolandic region, or on the theory that the contiguous parietal lobe was involved by the injury. In these cases, however, as in the numerous cases on record with anesthesia resulting from lesion in the Rolandic area it appeared that the one *constant* disorder whenever tested, was that of the stereognostic sense. That the Rolandic region is not, however, the sole seat of sensation is obvious; the theory that the stereognostic sense has its principal seat in the central gyri does not conflict, therefore, with the localization of the centers receiving and analysing the various cutaneous sensations in the postero-parietal convolutions, precuneus and gyrus fornicatus, for which strong evidence exists. Such evidence is epitomized in the recent text-book of Mills, and has been treated in detail in other communications by this author. It seems a reasonable supposition, however, that the Rolandic region contains at least the areas representing the principal aggregation of cutaneous and muscular memories whose correlated action acquaints us with the various qualities of external objects.

In view of the diametrical opposition of the opinions held by Dana and Mills respectively, and of the strong evidence adduced on both sides of the question, the problem seems to be one of harmonizing the clinical and experimental evidence rather than of confirming one of these views, which involves explaining away the evidence in favor of the other. We cannot accept the dictum of Dana that "it is hard to conceive of a sensory mechanism so complicated and clumsy as that required by a hypothesis of compound sensory centers, one in the limbic lobe and one in the motor cortex;" and if as he continues "no one has, I think, ventured to explain how such an anatomical arrangement could be put in harmony with the psychology of sensation," we are forced to find ourselves in the position of favoring some such complicated arrangement and of being sufficiently venturesome to hint at such explanation. It seems to us quite as logical to decide that these processes are complicated as to assume that they are simple.

In advocating his view of a sensory-motor memory organ in the Rolandic region, Dana has indicated an analogy between this area and Broca's convolution. If the clue furnished by this comparison is pursued to its ultimate conclusion, a theory is suggested which seems to reconcile the various apparently conflicting observations. It should be remembered in this connection that we need not hamper ourselves by an assumption that the orders of cerebral sensory neurones are limited in number.

Suppose we take for a working model the fairly established physiology of speech, we find the sensory side of this mechanism divided into several steps, each of which implies an interruption of the cortical sensory fibers with the interposition of a fresh neurone. If we consider, for example, the *visual* aspect of speech we find (1) a center for vision, sending fibers to (2) a center for so-called mind vision which in turn sends fibers not only in other directions but to (3) the assembling center in Broca's convolution known as that for the kinesthetic speech memories. From this center pass, finally, the stimuli calling into action the motor mechanism concerned in speech.

Turning now to the cutaneous sensory memories whose correlation plays so important a part in stimulating ordinary so-called voluntary movements, why may we not have similarly (1) the cortical structures receiving impressions of simple touch, temperature and pain, occupying mainly, or solely, as the case may be, the sensory regions of Ferrier, of Horsley and Schaefer and of Mills, let us assume (for diagrammatic purposes merely) the parietal lobe for the upper extremity, the gyrus fornicatus for the lower; higher in scale and perhaps more anteriorly, may be placed (2) the mechanisms, whose action represents the associative memories resulting in the appreciation of the character of the object felt; and finally why may there not exist (3) centers assembling the stimuli from the various groups of the second order and also from other sources to serve as the kinesthetic centers stimulating ordinary voluntary movements. In these centers the stereognostic sense should reach its highest development, and here should the mental image be formed which doubtless precedes every such movement, and which reproduces the memories of such sensations as will accompany the movement.

These centers would logically be placed most anteriorly, or nearest to those of so-called voluntary motion. The fact that the weight of evidence indicates that the fibers carrying muscle sense follow a separate course and terminate in the post-central gyrus seems to harmonize as well with this as with any theory advanced. Why may not the muscle sense fibers be conducted to the assembling centers by more direct paths than those of cutaneous sensations to play their part in the stereognostic function?

If some such scheme as we have outlined should be deemed reasonable, it would be easy to understand how lesions in the postero-parietal convolutions might also produce astereognosis through destruction of the centers of the second order or of the fibers passing from them, just as lesions impairing the condition of visual and auditory memories cause sensory aphasia?

Some such division of the cortical sensory areas would not only offer a logically reasonable compromise between the views of Dana and Mills, but it would follow an analogue already accepted, and seems on the whole fairly adaptable to the conflicting clinical reports submitted by the various students of this subject. Whatever view is adapted it is necessary to assume in capsular lesions with astereognosis, that though the touch sense is preserved, the fibers conveying cutaneous stimuli must be sufficiently impaired to preclude the perfect passage of *all* the impressions made on the skin by the object handled. This would obviously impair the sensory picture which must be analysed by the cortical centers, and thus interfere with the localizing, spacing, and posture senses? In attempting to analyze such cases it must be remembered that the classification adopted is arbitrary, convenient for practical examination and record, but not answering all physiological requirements.

To forestall the criticism that such a conception would too far simplify the processes of thought and action, it should be remembered that we have only sketched the possible course for one set out of the innumerable sets of fibers concerned. It is obvious that if such an assembling center exists, for example in the hand area of the post-central gyrus, it must be in connection with the speech memory centers, in order that the object may be named as well as recognised; again, the visual and auditory memories must have a representation here, and still further, the aggregation of sensory memory cells representing the hand must be connected, not only with the motor center for the hand, but also with those of other parts, for the exercise of the stereognostic sense in the hand may call out a movement elsewhere than in this extremity.

We have neither the training nor the desire to enter the domain of psychology, nor to localize the consciousness, which Loeb[18] defines as "a term for the phenomena determined by the mechanisms of association memories," a definition implying diffusion, not limitation. But notwithstanding this author's opposition to the theory of localization, clinical experiences supported by pathological reports seem to have established the fact that destruction of certain areas impair the mental processes in certain spheres. If this be true it seems not altogether unreasonable to suggest that possibly the association memories most intimately concerned in recognizing the feel of objects, have their principal seats in certain localities, the destruction of which will therefore especially impair the general mechanism of consciousness when brought to bear upon this particular function.

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[18] "Comparative Physiology of the Brain, and Comparative Psychology," G. P. Putnams Sons, N. Y., 1900.

An interesting case seen by one of the writers, (Dr. Walton), in consultation with Dr. C. M. Whitney, is suggestive in this connection.

Case V.—Mrs. B., 92 years of age, developed, about February 1, choreiform movements in the left arm, gradually spreading to the leg and face on the same side. At the time of consultation, twelve days later, movements were violent in the arm, moderate in the leg and marked in the face. The strength was not materially impaired in the affected members; the grasps were good; all voluntary movements were made, though in the inco-ordinate manner characteristic of chorea, especially marked in the arm. The temperature was 100, the pulse 160; the mind was clear. In the hand moderate astereognosis existed, though the touch was promptly felt. A bottle was called a purse, a fifty cent piece was called a copper, a square check was called round, but a knife was recognised promptly. Difficulty was experienced in comparing the sensation with that of the other hand, as that member was crippled by rheumatism; there was a difference, however, in favor of the right hand, as she here called the half dollar a "silver piece." In testing the sensation of the feet there was no bar to accurate comparison. The pencil applied transversely to the sole of the right foot was promptly recognized as a long object, but when it was applied to the left sole, the patient was unable to say whether she was touched by a point or a long object. Posture sense was lost in the toes on the left, space sense was markedly impaired, and localizing sense was extremely defective, the patient not once naming the toe touched, though she did this promptly on the right excepting once when she corrected herself directly. Touch, pain and temperature senses were preserved on the left as on the right. The choreiform movements were gradually replaced by a paretic condition, coma ensued, and death occurred six days later. There was no autopsy.

In this case the central convolutions were manifestly implicated by a lesion irritating but not destroying the motor area. While this case, like most of these adduced to support one or the other view regarding the cerebral localization of sensation, is susceptible of various interpretations, it requires no stretch of imagination to attribute the astereognosis to injury of the Rolandic area. The case is notable also as offering further illustration of astereognosis unaccompanied by motor paralysis.

It is significant that astereognosis has been present in every case of Rolandic disease coming under our observation since commencing this study. (Cases, 2, 3, 4, and 5.) It is true, as Mills[14] has stated, "that "innumerable cases have been reported of lesions of the motor cortex without the slightest impairment of sensibility," and this is one of the strongest arguments adduced against this region as the center for cutaneous sensation, but it is pertinent to enquire *whether the stereognostic sense was tested in these cases.* A certain number of cases of purely motor paralysis resulting from Rolandic lesion would not be incompatible with the view above outlined if the motor and stereognostic centers are distinct, but *innumerable* cases of this variety would certainly constitute a weighty argument against it. As an illustration of the variety of reports which should be excluded on account of failure to test the stereognostic sense we need only mention the recent, otherwise valuable, contribution of Hoppe.[15] Of the seven operations for brain tumor and cyst reported by this writer, four were performed upon lesions in the Rolandic region. In each of these four cases the sensation is reported normal, though three of the patients had complained of numbness. In only one of these cases, however, is mention made of the varieties of sensation tested, and in this one the report is limited to the senses of touch, pain and temperature.

In view of recent knowledge one is struck by the justice of Bastian's comment on *the difficulty in drawing conclusions regarding sensory judgments from experiments on animals.* We should certainly be chary of accepting Schaefer's[16] dictum that a single negative experiment on the brain of an animal eliminates the Rolandic region as a sensitive area, especially when he states that temporary sensory disturbance has sometimes appeared after subcortical section in this region, and that the animal does fail to remove the clip from the paralyzed limb after such operation. The latter phenomenon he explains by the absence of muscular twitching, and whatever sensory disturbance appears he attributes to edema or other local cause for the blunting of sensibility. If such hypotheses as these are accepted on the one hand, why not assume on the other that the failure to remove the clip represents something analogous to astereognosis?

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[14] "The Nervous System and Its Diseases." Lippencott, 1898, p. 341.

[15] Journal American Medical Association, February 2, 1901.

[16] Brain.

Even in the examination of hemiplegia resulting from infantile cerebral paralysis we are handicapped by the fact that many individuals suffering from congenital or infantile disease, have failed, through lack of use, to acquire the training essential to the development of the stereognostic sense.

This point has been well brought out by Claparéde[17] who reports the case of such a child with apparent astereognosis, who acquired the sense in the paralyzed hand after comparatively brief training.

In fact, we shall have to rely principally upon careful clinical observation of intelligent adult patients, and from such observations the facts doubtless will be gradually accumulated that shall place this localization on a par with that of speech and vision.

With regard to *the bearing of these observations on the seat of the lesion in hemiplegia*, while we are not in position to lay down final rules, it would seem that there are certain general indications which may help to guide us. In the first place, given a case of hemiplegia without astereognosis or other sensory disorder, the chances are perhaps in favor of capsular disease, though the supposition of separate centers would render theoretically possible purely motor paralysis from cortical disease in the Rolandic area. In point of fact, recent investigations indicate that purely motor hemiplegia is not so common as was formerly supposed, and that a large proportion of hemiplegics suffer from astereognosis (Verger found more than one-half in recent cases, Abba found 8% in fifty old and recent cases, Dercum found nearly half in forty-one cases).

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[17] Claparéde—Journal de Physiologie et de Pathologie Générale, September, 1899.

We have only had opportunity to study two (both probably capsular) fresh cases of hemiplegia since commencing this investigation, and of these two neither showed astereognosis. We have had opportunity, however, largely through the courtesy of Drs. Nichols and Howland, at the State Hospital, of Drs. Taylor and Lord at the Long Island Hospital, and of Drs. Lane and Noyes at the Boston Lunatic Asylum, to examine fifty-two old cases (varying in duration from two months to thirty years). Thirty-seven presented no trace of astereognosis or other form of anesthesia, but fifteen were unable to name objects and had lost space and posture, but preserved pain, touch and temperature senses. In several other cases the results were uncertain on account of aphasia or dementia. It has been claimed and is doubtless sometimes true that a long standing contracture with complete loss of motion in the fingers would prevent, from mechanical reasons, the recognition of objects;[18] this objection was guarded against by testing the posture, localizing and the spacing senses.

In point of fact, a number of cases with long standing contracture and immobility, in our cases, as in those of Dercum, answered promptly and correctly.

While the number of old hemiplegics so far examined is too few for statistical accuracy, our results, combined with the observations of others, would indicate that *a large percentage, perhaps a third or more of hemiphlegics show lasting astereognosis.* The distribution of the paralysis in most of our cases indicated capsular lesion, and it can be fairly assumed either that in such lesions involvement of the posterior limb is common, or that the sensory and motor fibers have no sharp dividing line, but are more or less mingled. The latter is the explanation offered by Dejerine.

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[18] It is interesting to note in this connection that Mills thus explains the apparent astereognosis in the case of Lloyd and Deaver. In this case Rolandic injury caused motor paralysis and inability to recognize objects in the hand, though touch, pain and temperature senses were normal. Test of the localizing, spacing and posture senses would have proved useful. In case the astereognosis resulted from sensory, not motor loss, the case would strongly suggest the central gyri for the seat of the stereognostic, but not for the cutaneous sensations. Single cases are only suggestive, however, never conclusive in this study.

In case of complete hemianesthesia involving the trunk the condition is apt to be one of hysteria either existing alone or superimposed on an organic lesion.

In case the so-called pseudo-hysterical form of anesthesia is present, that is, incomplete anesthesia of distal distribution and indefinite boundaries but including profound loss of touch, pain, and temperature senses, while we cannot always eliminate hysteria, we generally have to do with posterior capsular or thalamic lesion. This diagnosis will often be corroborated by the presence of hemianopsia. The studies of Verger, and those of Dejerine and Long would indicate that in the organic cases persistence of this form of anesthesia implies extensive involvement of the outer nucleus of the thalamus, the bulbo-thalamic, or the thalamo-cortical fibers.

Finally in such cases as those reported here, in which the astereognostic type of anesthesia is the initial or prominent symptom, we must remain in doubt whether the sensory fibres of the internal capsule or the cortical areas are primarily or solely involved, unless a monoplegic distribution, attacks of Jacksonian epilepsy, or other diagnostic features are added to the clinical picture.

The practical advantage of localising this function lies in its aid in selecting the seat of operation in case of suspected tumor, or other lesion susceptible of relief by surgical measures. In the light of practical results as well as of theoretical considerations it seems a safe working plan in operable cases with no other localizing symptom than astereognosis, to select for the center of the area to be exposed, a point in the ascending convolution at a height corresponding to the motor representation of the extremity involved. Such cases will doubtless be few in number compared to those of hemi- or mono-plegia in which astereognosis, added to the clinical picture, serves merely as a corroborative focal symptom.